efficiency of transport to hospital have a major influence on the outcome. The delayed effects of near-drowning on the lungs, however, mean that, whenever possible, people who appear to have survived such an incident should be observed in hospital where full facilities for intensive care are available.[8]

There are many other conditions where respiratory support may be necessary—as in septic shock or when the lung lesion is secondary to other disease—for instance, pancreatitis—or is iatrogenic—for instance overtransfusion. It should not be assumed, however, that the average patient in an intensive care unit has only a respiratory problem: cardiovascular, metabolic, renal, infective, and nutritional problems are all interlinked,[9] and attention to only one system is a sure recipe for disaster. In addition, there may be unique problems of communication with the patient and relatives, who may have to undergo a period of stress of a duration undreamt of 20 years ago.

Intensive care units are now a well-established and widely accepted part of the hospital scene. Over the years they have played a significant part in reducing mortality and morbidity in seriously ill patients by concentrating life-support equipment and skill and providing continuous monitoring of the patient's condition, both electronically and by the five (or six) senses of the attendant nurses. There is, however, an increasing need to know more about the patient and to know it continuously. Thus the future will see, in addition to developments in non-invasive monitoring, the increasing use of intravascular electrodes for continuous measurement of gas tensions and the more widespread use of pulmonary artery pressure measurements in assessing cardiac performance. The application of computing and data storage techniques to these and other variables enables derived values to be obtained immediately, thus allowing treatment to be based on a much greater appreciation of the extent of the physiological derangement.

Perhaps, above all, the intensive care unit sees no inter-disciplinary barriers in patient management. It can, and does, call on all specialties in the hospital when required, from the bacteriologist to the radiologist, and the biochemist to the pharmacist, as well as physicians, surgeons, and anaesthetists. In this way, through the organisation of the unit, the full resources of the hospital are brought to bear on the most critically ill patients.

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Contemporary Themes

Accidental poisoning deaths in British children 1958-77

NEIL C FRASER

Summary and conclusions

In the 20 years 1958-77 598 deaths were registered as due to accidental poisoning in British children under the age of 10—343 boys and 255 girls. Drugs caused 484 deaths, non-medicinal products 111, and plants three. The annual number of deaths reached a peak in 1964 but fell steadily thereafter; 16 deaths occurred in 1977. After 1970 tricyclic antidepressants replaced salicylates as the most commonly fatal poison. The next ten drugs most often recorded in 1970-7 were, in order, opiates (including diphenoxylate/atropine (Lomotil)), barbiturates, digoxin, orphenadrine (Disipal), quinine, potassium, iron, fenfluramine (Ponderax), antihistamines, and phenothiazines. In 20 years paracetamol caused one death, and before 1976 deaths caused by aspirin had fallen to fewer than two a year. Thus the introduction in 1976 and 1977 of safety packaging of these drugs can be expected to have little impact on the mortality from them in childhood.

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Introduction

Poisoning is a common accident of childhood,[1] but the outcome is rarely fatal.[2] In many cases little of the substance is actually swallowed and there may be uncertainty whether it will prove to be harmless or whether the ingestion should at once be taken seriously. The poisons that can prove fatal are neither those most frequently ingested nor those most often responsible for adult deaths. Most deaths in childhood are caused by a limited number of poisons, some of which are not commonly recognised as dangerous, while the toxicity of other substances is overestimated.

The number of deaths from accidental poisoning with solids or liquids has fluctuated considerably in the past 40 years (fig 1). The increase after 1935, which was attributed to changes in prescribing habits,[3] accelerated after the introduction of the NHS to a total of over 50 deaths a year in 1951 and 1952, when the principal causes were iron, strychnine, and antihistamines.[4] Strychnine was taken mainly in the form of Easton's syrup and tablets. A 60% reduction then occurred within five years but was followed by a further rise, to over 40 in 1964 and 1965. Since then numbers have again fallen considerably. In 1977 there were 16 deaths, the lowest figure since 1935. Deaths in this second "epidemic" from 1958 to 1977 have been analysed to identify the substances and trends. Some consequences of

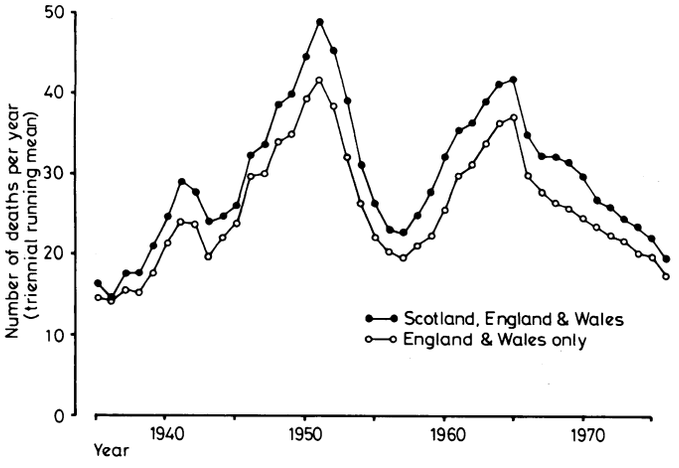

FIG 1—Accidental poisoning in children under 10 (Scotland, England, and Wales 1935-77). Number of deaths a year shown as triennial running mean.

preventive measures are apparent, and ways to reduce the current small number of deaths are suggested.

Sources of data

The numbers of accidental poisoning deaths in children under the age of 10 in categories E870 to E888 of the 1948 International Classification of Diseases (excluding poisoning by gases and vapours) were obtained from the annual reports of the Registrar-General for England and Wales and the Registrar-General for Scotland. Details of the substances concerned in the groups aged under 1, 1-4, and 5-9 were obtained for England and Wales for 1958-73 from the *Pharmaceutical Journal* and for 1974-7 from the Office of Population Censuses and Surveys. The corresponding details of Scottish deaths were obtained on inquiry to the Registrar-General for Scotland. In some cases the Registrar-General for England and Wales provided additional information, and in only six instances could the poisonous substance not be named.

Some deaths from accidental poisoning escape registration, either because a complication of the poisoning is regarded as the cause of death or because the poisoning goes unrecognised. A child who died at this hospital nine years ago was certified by the pathologist as "cot-death syndrome," although his history of respiratory failure after overdose of diphenoxylate/atropine (Lomotil) would now be recognised as characteristic.

Numbers of deaths

During 1958-77 598 deaths were registered as due to accidental poisoning in British children under 10 years of age. Less than one-fifth were caused by non-medicinal products. Ninety of the deaths were in Scotland. Forty-two children under 1 year and 44 aged 5-9 died. The male to female ratio was 1·3:1; under 5 years old it was 1·4:1. Death was registered as due to the ingestion of more than one substance in 15 cases (2% of the total); 9% of deaths resulted from compound preparations. Since the number of deaths in any year is small, figures have been grouped in five-year periods, 1958-62 (159 deaths), 1963-7 (186), 1968-72 (148), and 1973-7 (105). A change in the ratio of males to females may be observed—from 1·6:1 in 1958-62 to 1·1:1 in 1973-7—although the difference does not reach statistical significance.

Causes of death

Individual or related substances causing five or more deaths in 20 years are listed in the table. Medicinal and non-medicinal substances are ranked in order of overall frequency.

Medicinal

Twenty-three per cent of all deaths in the 20 years were caused by salicylates, mostly aspirin. Methyl salicylate was recorded on two occasions. Of the tricyclic antidepressants two-thirds were imipramine and one-sixth amitriptyline; seven others were recorded on 13 occasions, and five of the more recent deaths were due to formulations with phenothiazines. Barbiturate deaths were mostly due to hypnotic preparations. Phenobarbitone alone was recorded in three of the 45 cases. For over half of the deaths from iron the preparation was named as ferrous sulphate, and other formulations were specified in only five instances. Of the opiates and analogues, diphenoxylate (with atropine as Lomotil) and methadone (Physeptone) each caused ten deaths. Codeine, morphine, and dipipanone (with cyclizine as Diconal) each accounted for two. Digoxin includes other digitalis preparations.

Single drugs that caused two or three deaths in 20 years were methaqualone, chloral, dexamphetamine, hyoscine, and chlorpropamide. Most compound preparations are listed under the more toxic component—for instance, barbiturate and vitamins. Others include asthma preparations which, with varying combinations of barbiturate, opiate, antihistamine, antipyretic, xanthine, and adrenergic stimulant, accounted for five deaths. Two deaths each were caused by ergotamine and cyclizine (Migril) and by dicyclomine, doxylamine, and pyridoxine (Debendox).[5]

Non-medicinal

Lead caused one-third of deaths in the non-medicinal category, but the source of lead was seldom listed. The corrosives that caused death were caustic soda, cresol, phenol, and unspecified acids. Paraffin (kerosene) caused half of the deaths from petroleum products. Liquid polishes are included in this group. Ethylene glycol (antifreeze) probably caused four deaths and ethylene chlorohydrin, a solvent in photographic use that appears particularly toxic,[6] two. Three deaths were caused by cyanides and six by heavy metals other than lead. There was one death from a pesticide other than nicotine, fluoroacetamide, which is now in restricted use as a rodenticide. Single deaths were caused by ethylene dichloride (liquid glue), industrial hand cleaner, "paint," paraphenylenediamine (hair dye), polyglycol ethers, sodium chlorate, sodium nitrate, and soldering flux.

Changing pattern of fatal childhood poisoning

Within the overall rise and fall in poisoning deaths over 20 years there have been pronounced individual variations (table). While tricyclic antidepressants became established as the leading childhood poison of the 1970s, deaths from the most commonly fatal poisons of the forties, fifties, and sixties—strychnine, iron, and aspirin respectively—all showed a definite decline. Other poisons that became important in the later years (table) were orphenadrine,[7] fenfluramine,[8] and diphenoxylate,[9] and a few deaths from medicinal potassium[10] were also seen. Deaths from methadone, barbiturates, quinine, and digoxin continued, as did occasional deaths from antihistamines and phenothiazines.

Poisons that are rarely fatal

Although poisoning by paracetamol and dextropropoxyphene has caused increasing morbidity in adults,[11] no comparable problem exists in children.[12] Between 1958 and 1977 there was only one death attributed to paracetamol in a child under 10 and no death due to dextropropoxyphene.

The toxicity of household products in the quantities taken by children is still generally overestimated. Only one proprietary domestic product, a furniture polish, appears by name in the

20-year record. A single cosmetic is listed, "mascara containing lead," presumably surma.[13]

Plants are the most overrated poisons of childhood. In earlier decades there were occasional deaths, most caused by the umbelliferae (particularly hemlock water dropwort[14]) and the solanaceae (various nightshades[15]). From 1958 to 1977 there were three deaths, and in one the role of the ingestion in the child's demise is doubtful. The others were caused by hemlock and by *Amanita phalloides* (death cap), both in children aged 5-9. Laburnum is frequently cited[16] as the most toxic and commonly fatal poisonous plant in both children and adults, but there appears to be no report this century of a childhood poisoning death. One adult death in unusual circumstances has been recorded.[17]

Accidental poisoning deaths in children under 10. Scotland, England and Wales 1958-77

	No of deaths in each category per five-year period				
	1958–62	1963–7	1968–72	1973–7	Total
Medicinal:					
Salicylates	53	48	30	6	137
Tricyclic antidepressants	3	20	22	27	72
Barbiturates	13	12	13	7	45
Iron	11	23	7	2	43
Opiates and analogues*	4 (–)	6 (2)	6 (2)	10 (6)	26 (10)
Digoxin	5	6	7	5	23
Quinine	10	4	3	4	21
Strychnine	11	5	2	1	19
Orphenadrine	–	1	7	4	12
Antihistamines	3	3	3	2	11
Potassium (–thiazides)	–	2	2	4	8
Phenothiazines (alone)	2	3	1	2	8
Fenfluramine	–	–	5	1	6
Monoamine oxidase inhibitors	4	1	1	–	6
Phenylbutazone	2	2	–	1	5
Others	6	16	14	6	42
Total	127	152	123	82	484
Non-medicinal:					
Lead	10	16	7	6	39
Corrosives	5	4	2	5	16
Petroleum products	4	2	4	2	12
Paraquat	–	–	4	2	6
Alcohols	1	2	2	1	6
Nicotine	4	–	1	–	5
Others	7	9	5	6	27
Total	31	33	25	22	111
Plants	1	1	–	1	3

*Including diphenoxylate (numbers in parentheses).

Implications for prevention

As with self-poisoning in adults, the pattern of accidental poisoning in children is largely determined by the availability of prescribed drugs and by fashions in self-medication. This is most obviously illustrated by the 90% fall in salicylate deaths from 1964 to 1974 (fig 2). Paracetamol has been used increasingly as an alternative, and its present therapeutic popularity is reflected in its use in adult self-poisoning.[11] It is apparent that the introduction in 1976 and 1977 of safety packaging of aspirin and paracetamol[18] could have little effect on the already small number of deaths from aspirin and the negligible number in children from paracetamol. Severe therapeutic salicylate poisoning still occurs.[19 20]

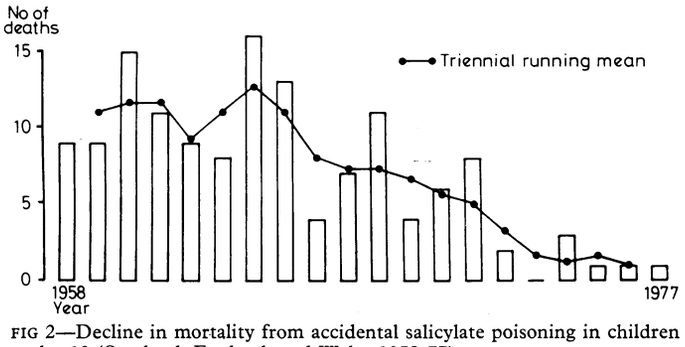

FIG 2—Decline in mortality from accidental salicylate poisoning in children under 10 (Scotland, England, and Wales 1958-77).

With increasing use of paracetamol (acetaminophen) in the United States, authorities there have expressed concern about its possible dangers in paediatric overdose.[21] British experience suggests that no serious problem will arise and that the number of deaths from salicylates should fall. Despite safety packaging there were 25 deaths from salicylates in children under 5 in the United States in 1976.[22]

A reduced number of ingestions may not necessarily lead to a fall in mortality. The introduction of child-resistant containers for aspirin, however, has led to fewer admissions to hospital,[18] and it may be hoped that a fall in mortality will also be seen should child-resistant containers be introduced for more important childhood poisons. Tricyclic antidepressants have since 1970 caused fatal childhood overdose most frequently and now account for one-third to one-half of deaths. It would thus have been more appropriate to introduce safety packaging of these drugs first, as was done in Australia.

By care in prescribing more could be done to reduce childhood as well as adult poisoning deaths. Barbiturates are unnecessary as hypnotics and should not be included in "tonics" or asthma preparations.[23] Strychnine should no longer be in medicinal use; a recent death was caused by Potensan, a combination of strychnine, amylobarbitone, and yohimbine (a supposed aphrodisiac). Since simple stretching of muscles at bedtime appears effective in preventing night-cramps, quinine might be prescribed less. Orphenadrine (Disipal) is now used principally to control the extrapyramidal side effects of phenothiazines. Perhaps in most cases benztropine could be substituted; it appears to have caused no deaths. Since fenfluramine (Ponderax) and other appetite suppressants have caused deaths, the short-term gains from their use should be weighed against this danger. Less toxic alternatives exist to diphenoxylate/atropine (Lomotil), which caused six known childhood deaths in 1973-7.

While some prescription of tricyclic antidepressants for adults is not questioned (and prescriptions for adults account for most childhood deaths[24]), they are still overused for enuresis, and their relative ineffectiveness compared with the enuresis alarm should not still have to be publicised.[1 25] Even less should these dangerous drugs be prescribed for "behaviour disorders" in young children since there is no convincing evidence of benefit.[26] While many of the family stresses associated with poisoning[27] cannot be anticipated, careful consideration should be given to prescribing any potentially dangerous drugs for the disturbed family.[28–30]

Early treatment of poisoning

Wider awareness of the lack of toxicity of many common substances ingested[31 32] should limit unnecessary treatment and hospital admission,[33] although emesis may sometimes be indicated to minimise unpleasant symptoms. On the other hand, knowledge of the poisons that can cause serious morbidity and death is necessary to recognise which ingestions need urgent attention.

Since several of the fatal poisons of childhood can be very rapid in onset of action, it is fortunate that most ingestions are discovered immediately. In some cases very early use of ipecacuanha emetic might prove lifesaving, and it should be available far more widely in the community.[34] The advantages of ipecac-induced emesis[35–37] need wider recognition in Britain. Adequate advice about its administration is seldom given, and many of the limitations or restrictions often placed on its use are not well founded.[38–40] The dangers of salt[41] and the ineffectiveness of mechanically induced vomiting[42] still need to be emphasised.

Possibility of iatrogenic death

Salt is only one inappropriate measure used to treat poisoning that can prove fatal. Inquiry into 36 childhood deaths reported to a Berlin poisons centre suggested that in at least five cases death had been caused by treatment rather than by the poisoning itself.[43] In several patients inappropriate fluid treatment had produced cerebral oedema, and it would be surprising if such deaths had never occurred in Britain. In salicylate poisoning, when forced diuresis does appear indicated, the syndrome of inappropriate antidiuretic hormone secretion may develop,[44] and forced infusion may be all that is carried out. A current recommendation[45] of 30 ml/kg/h, equivalent to seven litres in 24 hours for a 1-year-old child, is surely heroic, especially when no mention is made of these complications.

New poisons

Although most fatal poisons of childhood have been identified, other drugs in overdose may also cause serious illness,[46] and new ones will always appear. Attention has recently been drawn to the gap between the introduction of a drug and the application of effective measures to deal with its overdose.[47] For a drug that is not often taken there is also a considerable delay before its toxicity is widely recognised. As late as 1974, for example, a poisoning was reported[48] solely in terms of the tricyclic antidepressant taken, although the child had also ingested a potentially fatal dose of orphenadrine.

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Is there any risk in taking glyceryl trinitrate tablets after dental treatment during which the gums may have been damaged?

Possible complications include increased bleeding and local pain. Increased bleeding is a real but probably not a worrying risk because nitrates are principally venodilators, and although venous and capillary oozing might be more prominent there should be no risk from arterial bleeding; any tendency to the latter would also be offset by the reduction in arterial pressure. Local pain might be increased but the duration of action is quite short at about 20 minutes. Any possibility of a direct local toxic effect on the gums could be obviated by prescribing the nitrate as a paste applied to the skin in an occlusive dressing.

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What is the likely cause and meaning, if any, of persistent green stools in an otherwise healthy infant of 2 months who is breast-fed every four hours and whose average weight gain is 280 g a week?

During the early weeks of life the passage of green stools is a perfectly normal finding in fully breast-fed infants who are thriving well. The stools may become more green after being passed. Particularly if the stools have a normal *Lactobacillus bifidus* odour—and the doctor should check this personally—the mother may be completely reassured.